THE RADIOLOGIST AND EPIGASTRIC PAIN*

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IN THE PAST FEW YEARS abdominal radiology has advanced in many ways. Sharper images are now produced by faster film, faster screens and higher milliampere apparatus. Spot film devices allow visibly controlled angulation which may be altered from patient to patient. These devices also allow adjustable compression of underlying tissues. The result is a much more accurate assessment of tissue abnormalties. These modern techniques have had their application further improved by better tolerated opaques for gall bladder and kidney work. Barium emulsion is also improved, so that the barium does not drop out of suspension.

None of these advances however can replace skill in their use. Variation in method of study of each individual case must still be the responsibility of the conscientious, experienced radiologist. Abnormalities of many types cannot be recorded on films without his inquisitive fluoroscopic survey. Interpretation of his findings must be determined both by abnormalities of tissue structure and abnormalities of function.

All radiologists have experienced during the past fifteen years, the tendency of referring clinicians to ask, in effect "has this patient something organically wrong in his epigastrium or are his symptoms functional in origin?" By functional they evidently mean something either nonsurgical or existing in the patient's imagination only.

The above question requires answering certainly and marks an advance on the attitude of fifteen years earlier. The question at that time was, "this patient has a peptic ulcer. What chance has he of perforating, bleeding or obstructing?"

The time is coming and is now here for many clinicians to ask the right question of their radiological confrères. The question is very simple and puts the responsibility where the clinician really wants it placed. It is as follows: "This patient has pain or tenderness or discomfort in his upper abdomen. Will you try to find out for me the cause of the pain by your methods as I am not sure by my own?" The radiologist is then prepared to use any or all of his procedures to demonstrate abnormalities in any abdominal viscus. He may show functional abnormalities resulting from pathologic changes at some distance from the site of the symptoms.

The radiologist daily watches the œsophagus, stomach and duodenum function. By long observation of the normal he is taught to appreciate the abnormal. The frequency and amplitude of peristaltic action in normals is as easily and surely recognized by him as his office furniture would be. The observation of abnormalities of function or in other words functional abnormalities may be his clue to the solution of the symptom complex. The solution may be elusive and if not followed completely is unsatisfactory to the patient and clinician. It is discouraging to the radiologist. For him it is comparable to missing the last paragraph of a good murder mystery.

Some years ago we were prone to consider that if an ulcer crater or a space occupying new growth could not be demonstrated in a film we were examining a stomach which did not produce symptoms. Those days have gone forever.

We must always watch for the unusual. The multiplicity of unusual findings which can be demonstrated or strongly suspected in the upper abdomen forms a formidable group. Because of the bizarre pattern of their symptomatology these are the patients most frequently referred for x-ray studies. I am of the opinion that at least half of the patients referred for x-ray investigation of the gastro-intestinal tract and in which the more obvious lesions cannot be demonstrated will have demonstrable abnormalties of function. These demonstrable abnormalities are chiefly aperistalsis, hyperperistalsis, dilation of the lumen, spastic contractions of the lumen and marked variation in motility of the test meal. These findings are at variance with our usually appreciated causes for pain of intestinal origin. They are of course marked dilation or stretching of the bowel, tonic contractions of the bowel or ulcerative erosions of the mucosa anywhere in the gut.

We know that surgeons can enter the abdomen of a patient with infiltration anæsthesia of the wall only and cut, cauterize and suture without causing pain to the conscious patient. But traction must not be put on the mesentery. Actual palpation of an ulcer does not cause pain. When examining this patient in bed before surgery, a lot of dependence is placed on findings of deep tenderness or pain on deep pressure.

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Our physiologists have plotted referred pain areas from visceral pathology and have been very helpful. There seems to be only one explanation and that is, that the area of referred pain must also be receptive to stimuli or the pain cycle is broken. The radiologist, when screening, attempts as complete manipulation of gastric and intestinal contours as relaxation and costal margins will permit. His tender spot in duodenal ulcer is over the referred area and does not descend with the duodenum when the patient is changed from the horizontal to the upright position. This area is above and to the left of the demonstrable crater. In fact the structure immediately underlying it is most often the gastric lesser curvature. This does not mean that the patient has no sense of the origin of the pain. The intelligent co-operative patient will often tell you that pressure on the referred area causes pain elsewhere and will bring his pointing finger somewhere near a spot overlying the ulcer. You have all had the same experience with appendicitis. Pain may be classically placed and the appendix inches away from the expected location.

The more fixed viscera, notably the gall bladder and iliac loop of sigmoid, present tenderness more usually in the expected area. The latter of course is probably due to easier accessibility to palpation.

Let us now return to the epigastrium. The diagnosis of cardiospasm has long been considered a very simple radiologic problem. The massively dilated œsophagus containing food and with overhang on both sides of a pencil-sized constriction of the outlet is classical. Our endoscopists have made the discovery that cardiospasm is much more common than previously suspected. The early findings may be really difficult to demonstrate, as there may be a minimum of œsophageal stretching. It may be easily missed on a hurried fluoroscopic examination. This group now includes many patients with post-cricoid fullness due to neuromuscular derangement of the swallowing apparatus. Unusual delay in transit of the meal with even questionable dilation of the œsophagus where new growth, varices and ulcer can be ruled out, must be considered cardiospasm. The neuromuscular derangement may present another area of spasm well above the hiatus. These cases deserve repeat examinations, as peptic ulcer of the œsophagus may later be demonstrated in its lower segment.

Hiatal hernia, short œsophagus and partially intra-thoracic stomach, present such a distinctive finding that it should not be missed. The so-called sliding hernia with intermittent protrusion through the hiatus, present only in Trendelenberg position or on forced inspiration or on increased abdominal pressure is seldom productive of severe symptoms. Any hiatal hernia should therefore be examined for spontaneous reduction and proved to be either sliding or incarcerated.

Another group of uncomfortable people is the air swallowers. This group may have their abnormality recognized, their symptoms explained and their condition cured by the radiologist. We often see a half glass of barium cream enter the stomach with three or four times its volume of air. We watch the cardia expand and the left diaphragm ascend. The cure of course is to tell the patient what he is doing and where the supposed gas comes from. The second glass of barium is swallowed deliberately and in small mouthfuls with a minimum of air. The intelligent patient is immediately cured. Those who swallow air during their sleep are usually free of air before they reach the department and the diagnosis is made by elimination of other causes.

The next functional abnormality to be considered is gastrospasm. I find myself reporting this phenomenon much more frequently in recent years. By gastrospasm I do not mean an incisura at or below the level of a gastric ulcer. There is an occasional stomach usually of the oblique type, high up under the costal margin, which seems to fill completely with four to six ounces of the barium mixture. The mucosal folds are heavy and close together. These stomachs can only be studied in the horizontal position with a patient lying partially on his left side. The stomach empties rapidly. The presence of peristalsis rules out linitis plastica. For want of a better term I have considered this finding as indicative of gastritis.

Spasm of the pyloric segment of the stomach is a most difficult finding to assess. On fluoroscopy, peristalsis is interrupted two or three inches proximal to the pylorus and reappears within one-half inch of the sphincter. The mucosal pattern is compressed but not grossly deformed. The gastric profile in the area may suggest new growth. There is slight or no delay in emptying time. Localized pain is present which I believe has a diagnostic value in favour

of spasm rather than new growth. Fortunately the site is accessible to palpation and the absence of tumour usually can be verified. A differential diagnosis between pure spasm and antral gastritis is extremely difficult.

To differentiate between antral gastritis with spasm and mucosal distortion and early prepyloric new growth is sometimes difficult and often endoscopy is required. Where this is not easily available exploratory laparotomies have been done for visual diagnosis. Under general anæsthesia the stomach is relaxed even if inflamed and the suggestion is put forward that an occasional stomach should be studied, fluoroscopically, barium coated under general anæsthesia.

Pylorospasm in association with peptic ulcer within the canal or close to either extremity can present considerable difficulty. The difficulty is diminished and in many cases overcome by interrupting the examination. The patient is allowed to lie down relaxed for fifteen or twenty minutes. Actual visualization of the pyloric canal is essential for diagnostic accuracy. I have not had any help in this problem from the use of antispasmodics or sedatives.

Gastrospasm in association with duodenal ulcer or gall bladder or bile duct disease is sufficiently well known as to require mention only. The typical constricted crenated caput seen in duodenitis is classical and need not be further mentioned.

There is a large group of functional stomachs presenting antral spasm or pylorospasm of sufficient moment to cause upper abdominal symptoms, in which we later find abnormalities of the colon. These abnormalities may be appendicular or cæcal. They may be diverticula of the transverse colon or sigmoid. There may be the tapered cone-shaped rectal ampulla seen in fissure or thrombosed hæmorrhoids. In many of these I have noted that the upper abdominal discomfort is the predominant symptom. Many patients are averse to inviting a rectal examination and therefore do not mention anal or rectal symptoms unless severe.

In an effort to give these examinations practical value, I feel the radiologist should not have his examination limited by the clinician. Functional abnormalities of the stomach and duodenum should be followed by a complete gastrointestinal examination in a much greater number of cases. A rectal examination may provide the clue to treatment in many cases of functional dyspepsia.

CONCLUSION

The term functional dyspepsia has been applied in many cases of gastrospasm, demonstrable radiographically. In many of these, disease has been present elsewhere in the tract or its adnexa. I suggest that more complete x-ray examinations will solve the diagnostic problem in an increasingly greater number of so-called functional dyspeptics.

SCHISTOSOME DERMATITIS IN QUEBEC*

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DERMATITIS caused by schistosome cercariæ—more popularly known as "swimmer's itch", "water-itch" or "slough itch"—is known to be common in western Canada and in various parts of the United States as well as in Europe, Asia, and Africa. There have been few records from eastern North America and none from eastern Canada. The discovery of an infested area near Montreal was therefore of considerable interest, and the opportunity is taken to describe this infestation and to review briefly the epidemiology and symptomatology of the disease. The infestation in the Montreal region has apparently existed unsuspected for many years, and it is hoped that the present discussion will aid in the identification of other unrecognized areas of infestation.

The cercariæ which cause swimmer's itch are the larvæ of blood flukes similar in most respects to those which give rise to the human schistosomiases in various parts of the tropics and the Far East. The adults are minute, dioecious flatworms, which live in blood vessels—chiefly those of the intestine—of warm-blooded animals, either mammals or birds. There are many species of these worms, but the life cycle of all is similar in outline (Fig. 1). The female

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